A Topographic Event-Related Potential Analysis of the Attention Deficit for Auditory Processing in Aphasia

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McNeil (1982, 1988) and McNeil, Odell, and Tseng (1991) have hypothesized that the variability seen in aphasia may be due, at least in part, to a disorder in attention, effort allocation, or both. In a recent study, Peach, Newhoff, and Rubin (1993) employed electrophysiological testing procedures described by Naatanen (1990) to identify attentional deficits in auditory information processing following aphasia.

According to Naatanen (1990), a component referred to as the mismatch negativity (MMN) is elicited in event-related potentials (ERPs) when subjects are instructed to ignore auditory input consisting of standard and deviant tones (oddball paradigm) and attend to some distracting task such as reading or watching a video. The MMN component is extracted from a difference waveform produced by subtracting the standard stimulus ERP from the rare stimulus ERP (see Figure 1). This MMN component is said to reflect the subjects' automatic, preconscious attention to unattended auditory stimuli (Naatanen, 1990; Nyman et al., 1990).

In the study performed by Peach et al. (1993), aphasic subjects were tested under two conditions: ignoring and attending to auditory stimuli. The stimuli were composed of standard (frequent) and deviant (rare) tone bursts at 1000 and 2000 Hz, respectively. The MMN peaks for each subject were identified from the difference waveforms, and the peak latencies were then computed. Based on the pattern of latencies observed in the ignore condition, aphasic subjects were said to have demonstrated automatic attentional abilities similar to those of normal control subjects. The authors suggested, therefore, that aphasic subjects engage attention
Figure 1. Normal ERP waveforms in elderly control subject to standard 1000 Hz tones (thin line) and rare 2000 Hz tones (heavy line).

for auditory stimuli as normal listeners do. Performance in the attend condition, however, indicated that the aphasic subjects’ attention for completing discriminative tasks was deficient.

Recent investigations of the MMN have further defined this component in normals, in terms of its morphologic and topographic characteristics, and have provided some neurophysiological implications for these findings. Looking at the ignore conditions, Giard, Perrin, Pernier, and Bouchet (1990) identified a positive-going polarity in temporal regions below the Sylvian fissure accompanying the frontocentral negativity. Giard et al. (1990) and Paavilainen, Alho, Reinikainen, Sams, and Naatanen (1991) have suggested that these two components reflect a temporal generator located in the auditory cortex and a frontal generator that preferentially involves the right hemisphere. Giard et al. (1990) also suggested that the temporal component arising in the auditory cortex may be associated with a sensory memory trace and that the frontal component may represent an attention-switching process caused by the detection of a change at the sensory memory system.
These findings also have influenced the way in which MMN has been identified. Previously, the MMN has been measured at the peak of the negativity in the difference waveform corresponding to this component. Recognizing the contributions of the temporal cortex to the spatial characteristics of the MMN, Novak, Ritter, Vaughan, and Wiznitzer (1990) have reported an alternative approach that measures this component at the peak of its positive polarity in the temporolateral regions.

These findings led us to investigate more extensively the attentional mechanisms underlying auditory information processing in aphasia. To do this, we analyzed the morphologic and topographic characteristics of the MMN waveform elicited from aphasic subjects using the more recent methods of waveform identification described by Novak et al. (1990). The results were then compared to the findings obtained by Peach et al. (1993) when analyzing waveform latencies based on frontocentral negativities only. The following questions were addressed:

1. Are the morphologic characteristics (relative component amplitudes, onsets/offsets, time course) of the MMN waveforms found in the event-related potentials of aphasic subjects similar to those obtained in normal subjects?

2. What topographic patterns are associated with the MMN waveform in aphasic patients, and how are they related to those identified in normal subjects?

METHOD

Subjects

Seven subjects (five aphasic subjects and two controls) participated in this study (see Table 1). All subjects were native speakers of English and demonstrated normal hearing acuity for both ears in the frequency range between 500 and 2000 Hz. The aphasic group consisted of four males and one female ranging in age from 39 to 72 years. Aphasia was due to a single left hemisphere cerebrovascular accident for all subjects. Each aphasic subject was administered the Western Aphasia Battery (Kertesz, 1982) to obtain a measure of general language ability; the Aphasia Quotients for the group ranged from 88.9 to 14.4. The results were also used to determine the pattern of aphasia for each subject. Four patterns of aphasia were observed in this group, including anomic, Broca's, Wernicke's, and global aphasia. The time since onset of the aphasia ranged from 12 to 31 months.
Table 1. Subject Characteristics

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Sex</th>
<th>WAB AQ</th>
<th>Aphasia Type</th>
<th>Time Post Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MN</td>
<td>47</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>IG</td>
<td>71</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Aphasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EH</td>
<td>72</td>
<td>F</td>
<td>88.9</td>
<td>Anomia</td>
<td>24</td>
</tr>
<tr>
<td>PA</td>
<td>72</td>
<td>M</td>
<td>77.2</td>
<td>Broca</td>
<td>19</td>
</tr>
<tr>
<td>CS</td>
<td>72</td>
<td>M</td>
<td>49.8</td>
<td>Broca</td>
<td>15</td>
</tr>
<tr>
<td>AH</td>
<td>69</td>
<td>M</td>
<td>17.3</td>
<td>Wernicke</td>
<td>12</td>
</tr>
<tr>
<td>TL</td>
<td>39</td>
<td>M</td>
<td>14.4</td>
<td>Global</td>
<td>31</td>
</tr>
</tbody>
</table>

aWAB AQ = Western Aphasia Battery Aphasia Quotient. bTime post onset reported in months.

Instrumentation

Evoked potential testing and brain mapping was performed using the Biologic Brain Atlas Electrodiagnostic Testing System. Electrodes were placed using an ECI electro-cap electrode system. Twenty electrodes with impedances of less than 3 kΩ were placed according to the International 10–20 system. All electrodes were referenced to linked ears. The low- and high-frequency filters were set at 0.3 Hz and 100 Hz, respectively, and gain was 30,000 μV.

Data Collection

Auditory stimuli consisting of tone bursts were presented to each subject over Telephonics TDH-39P headphones at a rate of 0.8 per second. Each tone was characterized by a 20-ms plateau and a 20-ms rise/fall time. Standard and deviant stimuli consisting of 1000- and 2000-Hz tones, respectively, were presented randomly at a ratio of 5:1. Stimulus presentation continued until 50 samples were obtained to deviant tones. All samples were obtained using automatic artifact rejection.

The auditory stimuli were presented under a condition of passive attending. In this condition, subjects watched an inaudible segment from a familiar movie while the tone bursts were presented over headphones. Subjects were instructed to ignore the tones and to attend only to the movie. To increase attention to the movie and decrease attention to the tones, subjects were also told to be prepared to answer questions about the video.
Data Analysis

The morphologic characteristics of the MMN event were examined in this study by analyzing difference waveforms as suggested by Naatanen (1990). The difference waveforms were derived by subtracting the standard stimulus ERPs from the deviant stimulus ERPs. Two components of the MMN event were investigated: the temporal positivity arising from the auditory cortex and the frontal negativity arising from the right frontal cortex. To achieve this, the waveforms arising from two electrode sites, T5 and F4, were selected for analysis. These sites correspond to the left temporolateral region and to the right frontal convexity, respectively.

The following guidelines were used to analyze the waveforms obtained from each of these sites. To identify the temporolateral positivity associated with the MMN event, the waveform at T5 was first examined in the region between 70 and 250 ms, that is, in the area where the positive polarity is expected to occur (Giard et al., 1990; Nyman et al., 1990). The peak with the highest positive amplitude in this region that was also associated with frontal negativity at F4 was then selected and analyzed (Novak et al., 1990). MMN frontal negativity was identified subsequently by locating the greatest pattern of right frontal asymmetry at F4 following the temporolateral positivity at T5 and selecting the peak with the highest amplitude in this region of the waveform. Latencies and amplitudes for each of these peaks were then computed.

The topographic patterns associated with the MMN waveform were demonstrated in the brain maps constructed from the waveform amplitudes at each electrode site. The patterns observed in the aphasic subjects were compared with those of the control subjects, as well as with the expected patterns reported for normal subjects in the literature. Similarities and differences were noted and summarized descriptively.

RESULTS

Waveform Morphology

The morphological characteristics of the MMN difference waveforms at T5 and F4 are summarized in Table 2. All aphasic subjects demonstrated peak positive temporolateral latencies that were within the range both of the control subjects and also of normal subjects reported in the literature (70–250 ms). In fact, the latencies for the aphasic subjects were actually shorter in each case than those observed for either of the control subjects. The frontal latencies for the aphasic group were also similar to those of
Table 2. Peak Latencies and Amplitudes Associated With Temporal (T5) and Frontal (F4) Components of Mismatch Negativity Event

<table>
<thead>
<tr>
<th>Electrode Site</th>
<th>T5&lt;sup&gt;a&lt;/sup&gt;</th>
<th></th>
<th>F4&lt;sup&gt;b&lt;/sup&gt;</th>
<th></th>
<th>Latency Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ms</td>
<td>µV</td>
<td>ms</td>
<td>µV</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MN</td>
<td>128</td>
<td>0.65</td>
<td>132</td>
<td>−4.73</td>
<td>4</td>
</tr>
<tr>
<td>IG</td>
<td>126</td>
<td>1.87</td>
<td>148</td>
<td>−3.02</td>
<td>24</td>
</tr>
<tr>
<td>Aphasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EH</td>
<td>84</td>
<td>4.00</td>
<td>132</td>
<td>−1.55</td>
<td>48</td>
</tr>
<tr>
<td>PA</td>
<td>108</td>
<td>4.41</td>
<td>162</td>
<td>−1.63</td>
<td>54</td>
</tr>
<tr>
<td>CS</td>
<td>106</td>
<td>0.65</td>
<td>140</td>
<td>−0.89</td>
<td>34</td>
</tr>
<tr>
<td>AH</td>
<td>78</td>
<td>2.00</td>
<td>166</td>
<td>−1.75</td>
<td>88</td>
</tr>
<tr>
<td>TL</td>
<td>102</td>
<td>0.85</td>
<td>150</td>
<td>−0.61</td>
<td>48</td>
</tr>
</tbody>
</table>

<sup>a</sup>Left, posterior temporal region. <sup>b</sup>Right, frontal convexity.

the control subjects and within the reported ranges. These latencies either overlapped with or were slightly longer than the latencies observed in the control subjects. Large differences were observed, however, between the aphasic subjects and the control subjects in the amount of time between the onset of the peak positive temporolateral and peak negative frontal polarities. The younger control subject exhibited the expected pattern, consisting of a peak temporolateral positivity at T5 followed by a peak frontal negativity in close proximity at F4. The difference between the onset of these two events in this subject was 4 ms. In the older control subject, this difference increased to 24 ms, an effect that may be consistent with aging. For the aphasic subjects, though, the latency differences more than doubled to an average of 54.4 ms (SD = 20.2). These findings suggest that the aphasic subjects established a sensory trace of the deviant stimuli within normal limits but experienced abnormally long delays in switching their attention to these changes once they detected them.

Inspection of the amplitude data for these groups revealed a wide range of peak amplitudes for the temporolateral positivity at T5 in both the control and aphasic subjects. The amplitude for the F4 frontal negativity in the older normal (−3.02 µV) was less than that observed for the younger normal (−4.73 µV). This pattern is consistent with the age-related decreases in evoked potential amplitudes that have been reported consistently in the literature (Bashore, 1990). The F4 amplitudes for the aphasic subjects, however, were substantially below those observed in either of the control subjects. This decrease in amplitude suggests a reduction in the attentional resources allocated to the changes detected in the trace in sen-
sory memory. These findings also support the increased latencies observed between the previously described peak temporolateral positivity and frontal negativity. It appears that aphasic subjects required more time and allocated fewer attentional resources to the stimulus changes in this study than did either control subject.

**Topographic Patterns**

The topographic patterns of the aphasic subjects also showed distinct differences from the normal expected patterns. In normal subjects, the two components of the MMN event are associated with functional processes arising from two generators, one in the supratemporal plane of the auditory cortex and the other preferentially involving the right frontal region (Giard et al., 1990). The temporal generator underlies a neuronal mismatch process created by the deviant stimulus, whereas the frontal generator gives rise to an orientation to that mismatch. Our younger control subject exhibited the expected pattern of left temporal activity concurrent with bilateral frontal activity having right hemisphere preference. Temporal activity subsequently dissipated, leaving a strongly lateralized state in the frontal regions (see Figures 2 and 3). The older control subject initially displayed temporal activity with only minimal right frontal activity. This pattern did give way, however, to the bilateral frontal activity with right-sided preference that was observed in the younger control subject (see Figures 4–5).

The aphasic subjects produced several unexpected patterns. Two patients, EH and PA, demonstrated no frontal activity accompanying early left temporal activity. In addition, although right frontal asymmetry was observed subsequently, the amplitudes for these patients were significantly reduced in this region, as well as in all other cortical regions, relative to those of the control subjects (see Figures 6–9). Two other aphasic subjects, CS and AH, demonstrated a preference for left hemisphere frontal activity in conjunction with the activity of the temporal generator. For CS, the pattern associated with the frontal generator changed only with regard to the intensity of the associated amplitudes (see Figures 10 and 11). For AH, right frontal preference was established, but with persistent activity in the temporal region (see Figures 12 and 13). Finally, TL showed only right frontal activity in conjunction with the temporal activity corresponding to the mismatch process. This, of course, deviates from the expected pattern of bilateral frontal activity with a right hemisphere preference. As peak right frontal activity was established, temporal activity diminished as expected, but the amplitudes over the entire region were significantly reduced (see Figures 14 and 15). In fact, right frontal activity never exceeded that associated with the temporal mismatch process observed in the older control subject.
Figure 2. Difference waveforms and topographic pattern in younger control subject demonstrating left temporal activity with bilateral frontal activity having right hemisphere preference.

Figure 3. Strongly lateralized right frontal activity in younger control subject.
Figure 4. Temporal activity in elderly control subject with minimal right frontal activity.

Figure 5. Late bilateral frontal activity with right-sided preference in elderly control subject.
Figure 6. Early left temporal activity without accompanying frontal activity in aphasic subject EH.

Figure 7. Late right frontal activity with reduced amplitude in aphasic subject EH.
Figure 8. Early left temporal activity without accompanying frontal activity in aphasic subject PA.

Figure 9. Late right frontal activity with reduced amplitude in aphasic subject PA.
Figure 10. Left frontal preference in conjunction with activity of the temporal generator in aphasic subject CS.

Figure 11. Late maintenance of left frontal preference with reduced amplitudes in aphasic subject CS.
Figure 12. Left frontal preference in conjunction with activity of the temporal generator in aphasic subject AH.

Figure 13. Late right frontal preference with persistent temporal activity in aphasic subject AH.
Figure 14. Right frontal activity only in conjunction with temporal activity in aphasic subject TL.

Figure 15. Peak right frontal activity with reduced amplitudes in aphasic subject TL.
DISCUSSION

In this study, aphasic subjects demonstrated increased latencies between waveform peaks reflecting the temporal and frontal components of the mismatch negativity event. In addition, the peak waveform amplitudes associated with the cognitive processes underlying the MMN event were significantly reduced from those observed in control subjects. Together, these data indicate that these aphasic subjects required more time to allocate fewer attentional resources to the detection of changes in an auditory signal; as McNeil et al. (1991) would put it, the aphasic subjects expended less attentional effort per unit of time. In addition, the topographic patterns of the event-related potentials in these subjects demonstrate aberrations in the neural generators underlying these attentional processes. In sum, these findings are consistent with attentional deficits at the earliest, preconscious levels of attention.

McNeil et al. (1991) have suggested that some attentional deficits may result from an arousal impairment. According to these authors, "arousal subserves the ability of the organism to generate and allocate mental attention" (p. 32). Our current findings provide evidence that the attentional deficit in these aphasic subjects does not result simply from reduced arousal. The peak temporal latencies indicate the height of the timeframe within which our subjects detected and registered the change in the sensory stimulus. These latencies were well within the normal range; in fact, they preceded even those demonstrated by our control subjects. Had an arousal deficit been present, the aphasic subjects would have been expected to present longer temporal latencies than those of the control subjects, thereby indicating a difficulty in physiologically activating sensory systems to respond to the stimulus changes. Instead, the increased latency between detecting the stimulus changes and orienting to these changes, as indicated by the frontal latencies, is more consistent with a difficulty in effectively allocating attention to these changes.

The methodologic changes incorporated in this study produced results that contrast with those reported by Peach et al. (1993). Whereas those authors reported the attentional deficit following aphasia to be restricted to the completion of discriminative tasks, the present findings demonstrate that aphasic subjects do not engage attention for auditory stimuli as do normal listeners, even at the earliest, preconscious phases of orientation.

These findings do not provide a direct link between the attentional deficits observed in these aphasic subjects and their linguistic deficits, yet they do provide compelling evidence for an impairment in resource allocation that produced online processing deficits for auditory information. These deficits may, in turn, underlie some of the computational deficits associated with the language problems in aphasia. To address this issue, we are using evoked potentials to assess linguistic discriminations as a
means of further exploring the link between these attentional deficits and aphasic language. Perhaps the findings from work of this kind will provide the additional evidence needed to help determine how variations in attentional resources relate to the linguistic deficits observed in aphasia.

REFERENCES


